

U. S. DEPARTMENT OF LABOR

Employees' Compensation Appeals Board

---

In the Matter of ROGER L. DeSHONG and DEPARTMENT OF THE ARMY,  
LETTERKENNY ARMY DEPOT, Chambersburg, PA

*Docket No. 02-128; Submitted on the Record;  
Issued July 8, 2002*

---

DECISION and ORDER

Before DAVID S. GERSON, WILLIE T.C. THOMAS,  
A. PETER KANJORSKI

The issue is whether appellant has established that he sustained a respiratory condition in the performance of duty as alleged.

The Board finds that the case is not in posture for a decision due to a conflict in the medical evidence.

The procedural history of the case is as follows. On July 8, 1999 appellant, then a 54-year-old heavy mobile equipment metal mechanic, filed a Form CA-1 claim for an episode of acute shortness of breath sustained on June 29, 1999.<sup>1</sup> Appellant alleged that he experienced this attack due to occupational exposures to asbestos and other chemicals, resulting in asthma, interstitial fibrosis and asbestosis. On November 3, 1998 appellant filed an occupational disease claim for interstitial fibrosis (Form CA-2).<sup>2</sup> As of November 16, 1998, appellant was reassigned from the machine shop where he worked to an "office environment with minimal dust and dirt." Appellant stopped work in mid 1999.

By decision dated February 10, 2000, the Office denied the July 8, 1999 traumatic injury claim on the grounds that causal relationship was not established due to insufficient medical evidence. By decision dated January 17, 2001, the Office denied appellant's September 30, 2000

---

<sup>1</sup> A June 29, 1999 occupational health clinic note states that appellant was transported to the hospital by ambulance due to acute shortness of breath. In a June 29, 1999 report, Dr. G.E. Willwerth, an emergency room physician, noted that appellant presented with acute shortness of breath, with a history of occupational asthma and exposure to asbestos, abnormal pulmonary function tests and chronic obstructive pulmonary disease. Appellant's pulse oxygen was 96 and his respiration 28. Chest x-rays showed a seven centimeter density in the left lung base, thought to be a lipoma.

<sup>2</sup> The Office of Workers' Compensation Programs assigned the July 8, 1999 traumatic injury claim No. A3-0245533. The November 3, 1998 occupational disease claim was assigned No. A3-0238998. On November 20, 2000 the Office doubled the two claims under No. A3-0245533.

request for modification, but found that appellant had established a temporary, resolved aggravation of his symptoms for an unspecified period. Appellant again requested reconsideration by March 30, 2001 letter. The Office again denied modification by decision dated July 2, 2001.

By decision dated May 17, 1999, the Office denied the November 3, 1998 occupational disease claim on the grounds that causal relationship was not established, due to a lack of rationalized medical evidence. Appellant requested reconsideration on June 1, 1999 denied by merit decision dated August 31, 1999. Appellant submitted a January 10, 2000 request for reconsideration, enclosing an October 4, 1999 form report from Dr. Duane E. Sipes, an attending Board-certified family practitioner. By decision dated January 28, 2000, the Office denied reconsideration on the grounds that Dr. Sipes' report was repetitive of reports previously of record and, therefore, an insufficient basis, on which to reopen the case for a review on the merits.<sup>3</sup>

As the Office doubled the occupational disease and traumatic injury claims together on November 20, 2000 the evidence submitted under both claim numbers will be considered as a whole.

The Board finds that appellant submitted sufficient evidence to establish occupational exposure to toxic chemicals and asbestos, including several industrial hygiene reports prepared by the employing establishment regarding appellant's work and break areas.

Appellant's position description stated that metal workers were "constantly exposed to noise, dust, smoke, gases and dense and harmful volatile vapors and fumes," although safety equipment was provided to minimize exposure. In a November 16, 1998 letter, Fred Naessig, appellant's supervisor, confirmed that appellant performed grinding, sanding, welding, assembly, modification and repair on heavy military vehicles, including missile carriages.<sup>4</sup> Mr. Naessig stated that there was "a fair amount of dust when repair work is performed due to the disassembly process and the accumulation of dust in old equipment." Appellant wore an in-line air helmet as required.

In a September 16, 1999 report, Cheryl I. Hines, the employing establishment's industrial hygiene program manager, confirmed that in the machine shop where appellant worked, there were documented over exposures to lead, copper, iron oxide, aluminum, chromium, alum and formaldehyde.<sup>5</sup> Ms. Hines noted that appellant may have worn asbestos welding gloves from

---

<sup>3</sup> Appellant submitted a January 10, 2000 request, for reconsideration of the August 31, 1999 decision. In support of his request, he submitted an October 4, 1999 form report from Dr. Sipes, diagnosing restrictive and obstructive lung disease and checked a box "yes" indicating his support for causal relationship. Dr. Sipes commented that appellant was a "welder for many years -- chronic dust and metal particle exposure," with possible asbestos exposure. Dr. Sipes' October 4, 1999 report is highly repetitive of his reports previously of record, which also note appellant's history of occupational exposures and support causal relationship. This decision is not before the Board on the present appeal.

<sup>4</sup> Appellant worked on Hawk missile cabinets, Patriot missile trailers and Model 280 metal shelters.

<sup>5</sup> A March 8, 1989 industrial hygiene inventory showed that workers in appellant's shop were exposed to adhesives containing toluene, solvents containing acetic acid, 1,1,1, trichlorethane, methyl ethyl ketone, methyl

1985 to 1989 and the vehicles on which he worked had “asbestos brake linings and asbestos gaskets on engines and exhaust systems.” She noted that pipe insulation containing 10 to 20 percent amosite asbestos and 5 to 10 percent chrysotile asbestos had fallen from the ceiling to the floor in the building where appellant worked. Ms. Hines stated that bondo repairs, using an acetylene torch and welding involved exposures to sealer dusts, titanium dioxide, aluminum, lead, chromium, zinc, barium, calcium, cadmium, copper, iron, iron oxide, magnesium, manganese, molybdenum, nickel, sodium and formaldehyde. She provided the following list of chemicals present in the seals and glues appellant used: “[t]itanium dioxide, magnesium, toluene, calcium carbonate, methyl ethyl ketone, methyl isobutyl ketone, rosin acids, acrylonitrile, tri (butoxyethyl) phosphate, calcium aluminum silicate, cyclohexane, n-hexane, isobutene, dimethyl ether, propane, vinyl chloride/vinyl acetate polymer, polyurethane polymer, kaolin, carbon clack, epoxy resin, n-butyl glyceridyl ether, benzoyl peroxide, butyl benzyl phthalate, styrene monoxide, aniline, butyl acetate, phenolic resin (formaldehyde), manganese dioxide, polysulfide polymer, dihydroxy polydimethyl silicane, crystalline silica, polydimethyl siloxane, amorphous silica, alkytrizacetoxysilicanes, acetic acid, iron oxide, zinc oxide and salicylic acid.”

The Board finds that appellant has also submitted sufficient medical evidence to substantiate the presence of interstitial, fibrotic lung disease and chronic obstructive pulmonary disease, including objective test results. Annual pulmonary function tests and spirometry studies performed from June 11, 1986 to July 24, 1998 showed progressive mild restrictive and obstructive airway disease, with progressively reduced diffusion capacity. A May 21, 1996 chest x-ray showed interstitial fibrosis and early chronic obstructive airway disease. July 24, 1998 x-rays showed “calcified granulomas in right hilum,” a possible mass in the left posterior sulcus and “minimal accentuation of interstitial markings ... [which] may be compatible with clinically suspected interstitial fibrosis.”

---

busobutyl ketone and fillers containing ground asbestos. A March 26, 1996 industrial hygiene report showed the presence of the following substances, classified as hazardous: “respirable dust,” titanium dioxide, aluminum, lead, chromium, zinc, barium, cadmium, calcium, nickel, sodium, magnesium, manganese and dust from sanding Bondo plastic body compound. A February 20, 1990 industrial hygiene survey demonstrated that employees in appellant’s shop had been overexposed to lead and possibly overexposed to copper and iron oxide. Additional safety equipment was installed to reduce toxic exposures. An April 17, 1990 industrial hygiene survey found toxic dust coating the tables in the metal shop break area, including: aluminum, barium, calcium, cadmium, chromium, copper, iron, magnesium, manganese, molybdenum, nickel, lead, sodium and zinc. The industrial hygienist reported that the tables were “coated with dust,” and employees were eating and drinking at these tables. A July 12, 1991 industrial hygiene survey found high levels of lead, aluminum, chromium and cadmium in vehicle and “cabinet” paint tested. It was determined that cutting through these paints with an acetylene torch, which was one of appellant’s duties, would cause nausea and a burning sensation in the eyes, lungs and throat as had been reported by several employees. An April 24, 1997 industrial hygiene chemical inventory found that appellant and his coworkers in the machine and body shop were exposed to Rexcel coatings, Technit conductive system epoxy, silver conductive epoxy, aluminum epoxy, sealants, Bondo resins, gold alodine, contact cement, caulking, alum tap, hardeners, solders, aircraft paint remover, spray adhesives and structural adhesives. An October 19, 1998 industrial hygiene survey showed the pipe insulation in the vehicle shop had fallen when employees performed a “self-help ventilation duct installation project,” with the insulation composed of 10 to 20 percent amosite asbestos and 5 to 10 percent chrysotile asbestos. Air particles were determined to be within the acceptable limit of .01 fibers per cubic centimeter of air.

In a December 9, 1998 report, Dr. Henry K. Smith, a Board-certified radiologist and certified B reader,<sup>6</sup> found that chest x-rays showed “mild increased markings and interstitial changes ... most pronounced in the mid to lower zones of classification S/T, 1/0 and consistent with early mild asbestosis.”

Appellant submitted reports from July 16, 1998 to May 25, 1999 from Dr. Johny P. Alencherry, an attending Board-certified internist specializing in pulmonology. Dr. Alencherry noted that appellant’s shortness of breath, his 40 year cigarette smoking history ending in 1998 and that he worked “in metal fabricating and is exposed to dust, fumes, copper and nickel,” as well as “some exposure to asbestos.” He found that pulmonary function testing showed a reduced forced vital capacity correlating with a possible underlying restrictive airway disease process, as well as mild bronchial hyperactivity on methacholine challenge.<sup>7</sup> Dr. Alencherry diagnosed minimal chronic obstructive pulmonary disease, lung fibrosis, an “element of occupational asthma,” and small airway disease possibly related to cigarette smoking. He opined that these conditions were caused or aggravated by appellant’s “occupational exposure to dust and fumes....” Dr. Alencherry recommended that appellant change his work environment to one without dust or fumes.

Appellant also submitted reports from December 17, 1998 to June 9, 2000 from Dr. Sipes, an attending Board-certified family practitioner. He noted that appellant’s increasing shortness of breath, during the past several years, with several pulmonary illnesses since 1987. Dr. Sipes diagnosed stationary chronic obstructive pulmonary disease and progressive fibrotic, “restrictive lung disease correlating with asbestosis (mild).” He opined that the asbestosis was objectively evident on May 21, 1998 pulmonary function testing and in December 29, 1998 x-rays “showed mild interstitial fibrosis in the mid to lower lung fields consistent with early mild asbestosis.” Dr. Sipes stated that occupational exposures caused “mild emphysema and had aggravated appellant’s condition, as “asbestos and dust exposure exacerbates COPD [chronic obstructive pulmonary disease]” and caused more rapid progression of restrictive lung disease.

Appellant also submitted a March 23, 2000 report from Dr. John B. Paulus, an attending Board-certified internist specializing in pulmonology. Dr. Paulus reviewed medical records and provided a detailed history of occupational chemical and asbestos exposures, smoking and treatment provided. On examination he found “bibasilar crackles with decreased respiratory excursions,” correlated on pulmonary function tests obtained that day showing “decreased CO [carbon monoxide] diffusion as well as restrictive disease.” Dr. Paulus opined that “[a]fter a latency period consistent with pulmonary asbestosis, [appellant] now complains of shortness of breath” limiting activities of daily living. He found that appellant’s chest x-rays “confirm[ed] the clinical diagnosis of pulmonary asbestosis,” demonstrated by the bibasilar crackles and decreased respiratory excursions.

---

<sup>6</sup> A “B reader” is a radiologist who undergoes specialized training, examination and certification by National Institute of Occupational Safety and Health (NIOSH) in the early detection of asbestosis and pneumoconiosis.

<sup>7</sup> A December 23, 1998 pulmonary function test showed “[m]ild airway obstruction, predominantly small airway dysfunction,” a “[m]oderate restrictive lung process,” “[s]ubstantially reduced diffusion capacity,” “[s]ignificant respiratory alkalosis with metabolic acidosis and mild hypoxemia” suggestive of pulmonary fibrosis.

The Office referred appellant for a second opinion examination to Dr. Jose Acosta, a Board-certified pulmonologist. In a December 20, 2000 report, Dr. Acosta provided a history of injury and treatment and reviewed the medical record. He diagnosed chronic bronchitis, related to smoking. Dr. Acosta noted that appellant's occupational exposure to dust and fumes may have contributed to his shortness of breath and bronchitis, but that the airway disease was more likely related to smoking. He ordered pulmonary function tests and a high resolution computerized tomography (CT) scan to "clarify better the presence of interstitial lung disease."

Dr. Acosta obtained a December 28, 2000 pulmonary function test and exercise test, which he opined were within normal limits. December 20, 2000 x-rays showed "mild chronic appearing interstitial prominence." A December 28, 2000 CT scan showed a large fat containing diaphragmatic hernia in the left lower lobe and "slightly prominent parenchymal interstitium, which may reflect a chronic bronchiectatic type process." Based on these test results Dr. Acosta stated that, in a January 3, 2001 report, appellant had "bronchial asthma and/or chronic bronchitis," with no significant pulmonary fibrosis or pleural plaque suggestive of asbestosis. He opined that these findings were due to smoking, but that exposure to dust and fumes may have aggravated appellant's asthma." In a January 11, 2001 report, Dr. Acosta stated that appellant sustained a temporary aggravation of his pulmonary conditions due to occupational exposure to dust and fumes and that such aggravation ceased when the exposures ceased in 1999 when he stopped work.

To establish that an injury was sustained in the performance of duty in an occupational disease claim, a claimant must submit the following: (1) medical evidence establishing the presence or existence of the disease or condition for which compensation is claimed; (2) a factual statement identifying employment factors alleged to have caused or contributed to the presence or occurrence of the disease or condition; and (3) medical evidence establishing that the diagnosed condition is causally related to the employment factors identified by the claimant. The medical opinion must be one of reasonable medical certainty and must be supported by medical rationale explaining the nature of the relationship between the diagnosed condition and the specific employment factors identified by the claimant.<sup>8</sup>

The Board finds that in this case, causal relationship cannot yet be determined dispositively as there is a conflict of medical opinion between Dr. Acosta, for the government and Drs. Paulus, Alencherry and Sipes, for appellant. Dr. Acosta opined that appellant had no findings indicative of asbestosis and that his occupational exposures caused a temporary, resolved aggravation of his pulmonary disease. However, Drs. Paulus, Alencherry and Sipes found that appellant had objective evidence of fibrotic interstitial disease indicative of asbestosis, as demonstrated by pulmonary function testing, chest x-rays and findings on auscultation. Appellant's physicians also opined that occupational exposures to dust, fumes and asbestos permanently accelerated his fibrotic lung disease and aggravated his chronic obstructive pulmonary disease.

Where there is a conflict of medical opinion between a physician making an examination for the government and the employee's physician, the Office must appoint a third physician to

---

<sup>8</sup> *Charles E. Burke*, 47 ECAB 185 (1995).

conduct an impartial medical examination to resolve the conflict.<sup>9</sup> As there is a clear conflict of medical opinion in this case, the case must be remanded to the Office for further development to resolve the conflict.

On remand of the case the Office will refer appellant, the medical record and a statement of accepted facts, revised to include all chemical exposures listed by the employing establishment, to an appropriate Board-certified specialist or specialists, to obtain a rationalized opinion regarding the causal relationship of appellant's occupational exposures and any pulmonary or medical condition. The Office shall also obtain specific opinion regarding the periods of any occupationally-related aggravation of appellant's obstructive pulmonary disease as accepted by the Office in its January 17, 2001 decision and pay any compensation due and owing for those periods.

In obtaining the impartial medical opinion, as in previous development of the medical evidence, the specialist appointed will have to address appellant's 40-year history of smoking one pack of cigarettes per day. However, the Board notes that an employee is not required to prove that occupational factors are the sole cause of his claimed condition. If work-related exposures caused, aggravated or accelerate appellant's pulmonary condition, appellant is entitled to compensation.<sup>10</sup> The appointed specialist or specialists should provide a detailed explanation differentiating between the causes of the obstructive disease attributed to cigarette smoking and the interstitial, restrictive lung disease caused, aggravated or accelerated by occupational chemical and asbestos exposures. Following this and any other development deemed necessary, the Office shall issue an appropriate decision in the case.

---

<sup>9</sup> 5 U.S.C. § 8123(a).

<sup>10</sup> *Beth P. Chaput*, 37 ECAB 158 (1985).

The July 2 and January 17, 2001 decisions of the Office of Workers' Compensation Programs are hereby set aside and the case remanded for further development consistent with this decision and order.

Dated, Washington, DC  
July 8, 2002

David S. Gerson  
Alternate Member

Willie T.C. Thomas  
Alternate Member

A. Peter Kanjorski  
Alternate Member